Managing Acid Base and Electrolyte Emergencies in Austere Conditions

(ie, the wards and clinics)

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What I'll talk about

- Metabolic acidosis
- Metabolic alkalosis
- Hyponatremia
- Hypernatremia
- Hyperkalemia
- Hypokalemia

So the patient has a metabolic acidosis--then what?

Acidosis

- Is it JUST a metabolic acidosis? Use the Winter's formula (or your trusty acid base nomogram) to check.
- Winter's formula-- $1.5*HCO3+8=\text{expected } PCO2$
- PCO2's above or below these values indicate superimposed respiratory acidosis and alkalosis, respectively

Acidosis

- So what kind of metabolic acidosis is it?
- Calculate anion gap --check the values at your hospital, they may not be the same as at WRAMC (which is supposed to be 12)

Acidosis

- Anion Gap Acidoses:

  Ketoacidosis (both diabetic and alcoholic)

  Lactic acidosis (from underperfusion and overproduction, liver disease)

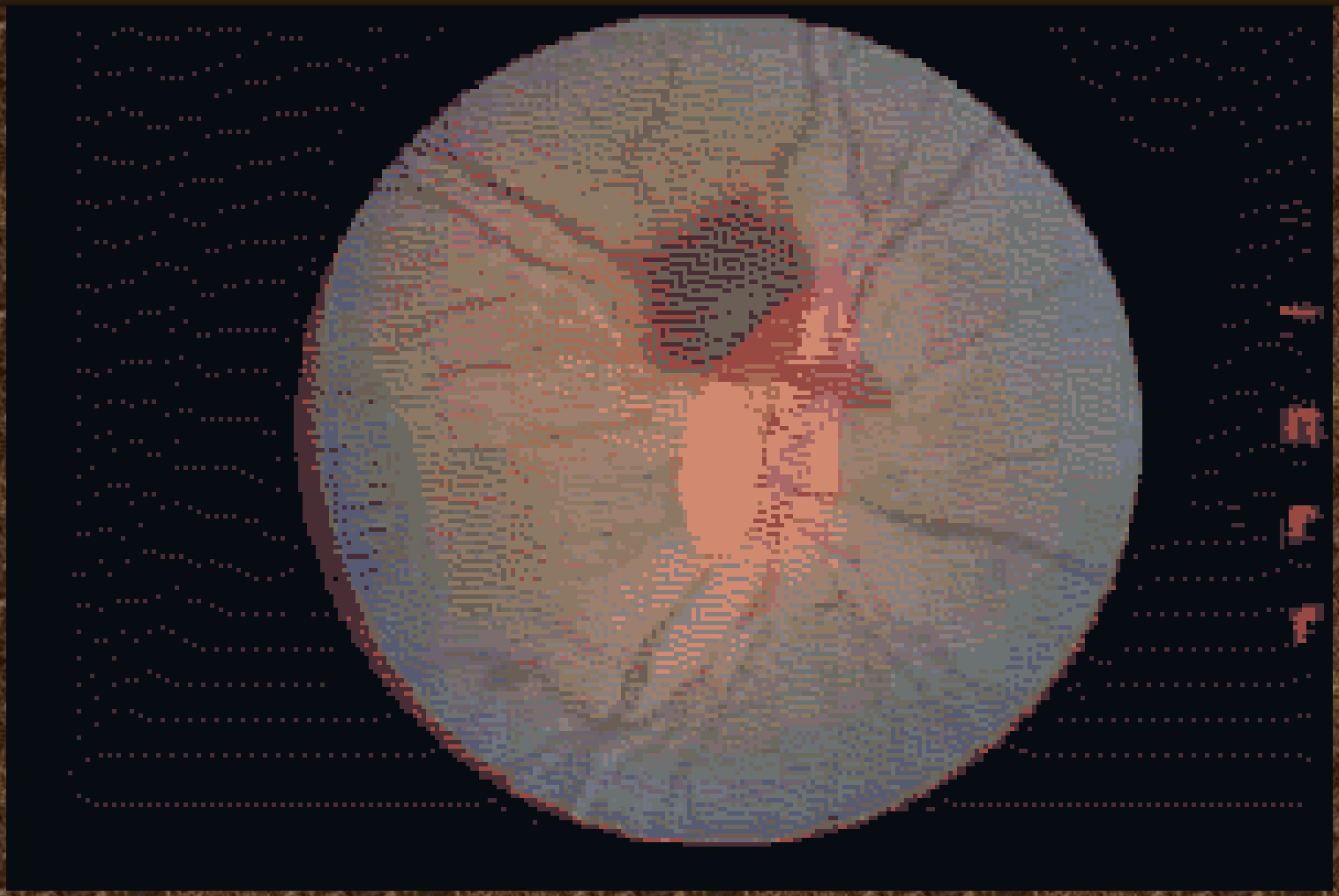
  renal failure

  toxins, esp. aspirin, alcohols (methanol, ethylene glycol)

  Consult your trusty blue book (“Facts and Formulas” by Robert C. Rollings) for the laundry list

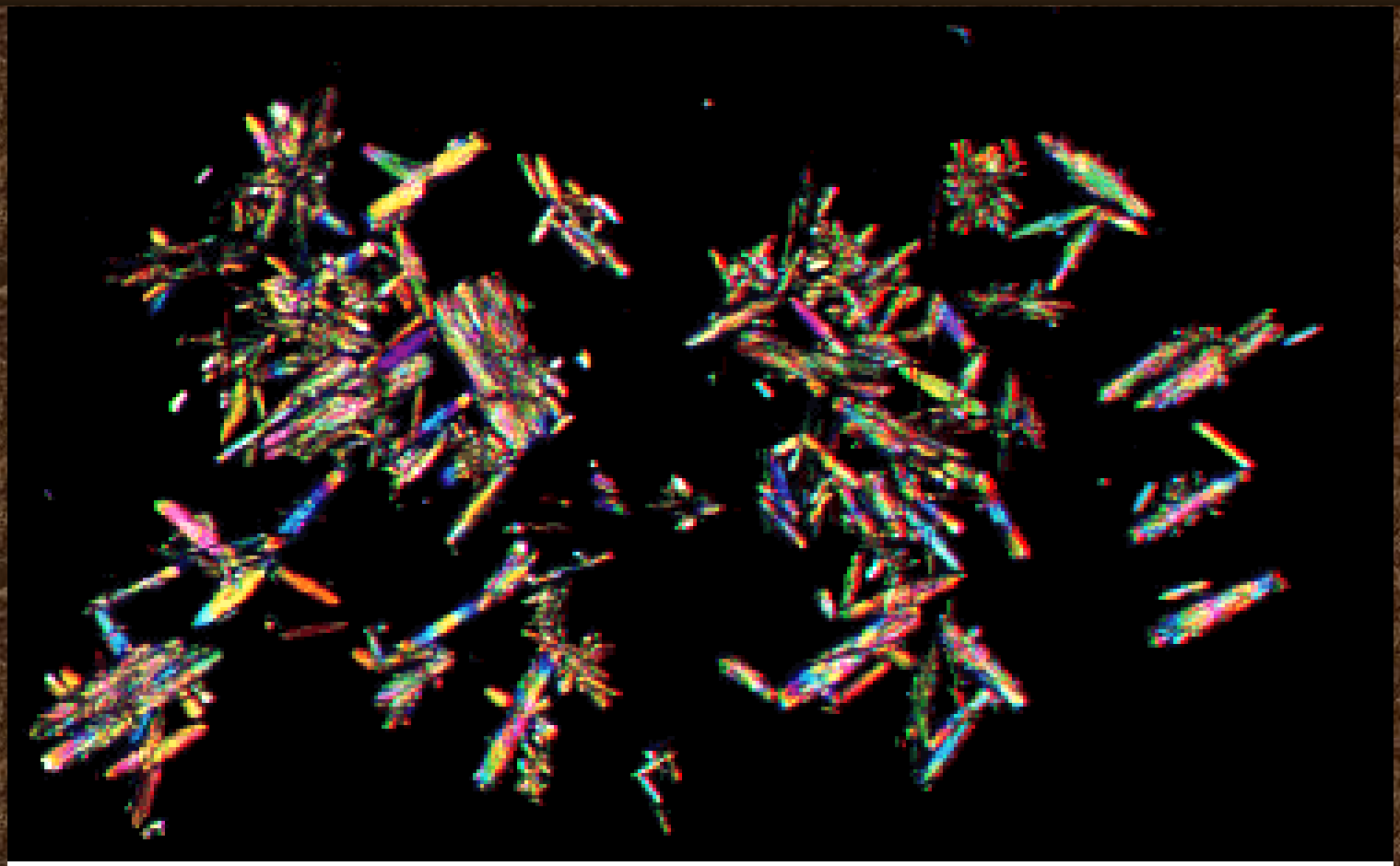
Acidosis

- Remember to look at the fundi for papilledema and retinal hemorrhages in METHANOL toxicity
- Look in the urine for calcium oxalate crystals (look like cubes--THE BORG) in ETHYLENE GLYCOL toxicity
- Check a serum osmolar gap--measured serum osmolality minus calculated serum Osm
- 2*Na+Glucose/18+Bun/2.8



Fresh retinal hemorrhage from:
http://www.cmsp.com/im0438.htm

Calcium oxalate crystals Urine sediment showing both dumbbell-shaped calcium oxalate monohydrate (long arrow) and envelope-shaped calcium oxalate dihydrate (short arrows) crystals. Although not shown, the monohydrate crystals may also have a needle-shaped appearance. The formation of calcium oxalate crystals is independent of the urine pH. Courtesy of Frances Andrus, BA, Victoria Hospital, London, Ontario.



Calcium oxalate monohydrate crystals Urine sediment viewed under polarized light showing coarse, needle-shaped calcium oxalate monohydrate crystals. These crystals have a similar appearance to hippurate crystals. Courtesy of W Merrill Hicks, MD.

Guide to toxins

AGAP	Osmolal GAP	Toxin
Nl	↑	Isopropyl alcohol
↑	Nl	Salicylate, cyanide
↑	↑	Ethylene glycol, ETOH, methanol

Estimating Drug Levels

- Drug concentration=osmolar gap *(Molecular weight of toxin/10)

Acidosis

- Non-Anion Gap Acidoses
- Stool loss most common--esp cirrhotic pt treated with lactulose
- RTA (with low potassium except for type IV RTA, usu due to diabetes mellitus)
- Diamox
- Toxins such as Toluene
- Ureteral-sigmoid diversion
- partial or complete urinary tract obstruction (with high potassium)
- cyclosporine (with high potassium)

Acidosis

- How do tell between a GI and a renal cause of RTA?
- Check urine electrolytes--in diarrhea, pt. usually volume depleted and Urine Na <20
- Check a urine anion gap
- The urine chloride should be 20 or more than the sum of the NA and K in a significant acidosis (~pH 7.3)
- If not, may be renal cause EXCEPT:
- UNa must be >20
- Cannot have a coexisting anion gap acidosis
- Urine Osm must be > serum Osm

Urine Anion Gap

- The urine anion gap is most sensitive for distal RTA
- Proximal RTA's may have a preserved urine anion gap.
- Distal RTA--urine pH >5.5 on a FRESH urine sample even with severe acidosis
- Proximal RTA--urine acidification intact.
- Can distinguish them by Fractional Excretion of bicarbonate WHEN SERUM BICARBONATE CORRECTED (Proximal RTA >15%, Distal RTA <5%). This is usually not an emergency and won't be discussed further.

Acidosis

- Can you have both an anion gap and a non-anion gap acidosis?
- yes! So you're not done--calculate a Delta AGAP and Delta HCO3
- They should be within three of each other.
- If Delta AGAP is greater than Delta HCO3, could be Lactic Acidosis or a preexisting metabolic alkalosis
- If Delta HCO3 is greater than Delta AGAP, could be coexisting non-anion gap acidosis--but can't use urine anion gap.
- The importance of checking this is to broaden your differential diagnosis and to alert you to ask for more history

Hybrid Metabolic Acidosis

- You can't use a urine anion gap here, but you can use a urine osmolar gap
- Calculated urine Osm = 2*(Na+K) +UUN/2.8+ Glucose/18 (if glycosuria on dipstick)
- An osmolar gap of <20 indicates impaired renal ammoniagensis

Acidosis

Key difference in treatment of AGAP and Non-AGAP acidosis

- For AGAP acidosis, you wouldn't use HCO3 therapy until pH <7.2 or 7.1
- For Non-AGAP acidosis, the primary problem is insufficient HCO3, not overproduction of acid, so you WOULD use HCO3 therapy to correct serum HCO3 to about 20.

Metabolic Alkalosis

- Use the urine Chloride--20 is the cutoff
- Urine sodiums may be falsely elevated in alkalosis because they are being excreted with bicarbonate

Alkalosis

- Chloride sensitive alkalosis (Urine Chloride <20)
- upper GI loss (vomiting, NG suction)
- "Contraction alkalosis"--due to chloride loss from diuretics. Isotonic volume depletion is NOT associated with metabolic alkalosis
- Responds to saline--NOT Diamox

Alkalosis

- Chloride Resistant Alkalosis (Urine Chloride >20)
- Most commonly due to Hypokalemia (if it developed as an inpatient)
- Divided into hypertensive and non-hypertensive, almost always with hypokalemia.

Chloride Resistant Metabolic Alkalosis

- Hypertensive
- Hyperaldosteronism (can be bilateral adrenal hyperplasia or adrenal adenoma)
- Cushing's Syndrome (or disease)
- Apparent Mineralocorticoid excess (consult Renal and/or Endocrine at this point, and probably for all the above too)
- Liddle's syndrome (a primary renal disease and a diagnosis of exclusion)

Chloride Resistant Metabolic Alkalosis

- Normotensive
- Bartters syndrome (a primary renal potassium wasting disorder with impaired bicarbonate metabolism)
- Pseudo Bartters syndrome (from vomiting or diuretic use)--much more common than real Bartters, look for stains/eroded enamel in the back of front teeth, hints of diuretic use. If you admit them to the hospital they will convert to Chloride SENSITIVE metabolic alkalosis.
- Diuretic phase of ATN

Electrolytes

Hyponatremia

Hyponatremia

- The most common inpatient electrolyte disorder
- OK, we know about fluid restriction (1.0 L or less) and saline administration if they're dry
- We want to know about HOT SALT!
- Don't forget the serum and urine osms and urine lytes with creatinine first
- Consider adrenal insufficiency if they're hypotensive and the K is not low

Hyponatremia

- Who gets it?
- Patients with acute (<48 hrs onset) hyponatremia
- Those with symptoms (seizures, otherwise unexplained mental status changes)
- ESPECIALLY premenopausal females

Hyponatremia

- ACUTE SYMPTOMATIC HYPONATREMIA
- Raise Plasma sodium 1.5-2.0 meq/hr for the first 3-4 hrs, then max of 12 meq per 24 hrs
- Calculate the sodium deficit:
- 0.5 (in females)*wt (kg)*(125-serum sodium)=total meq sodium needed over 24 hrs.
- 0.5*wt(kg)*8 (2 meq/hr over 4 hrs)=total meq sodium needed over first 4 hours.

Hyponatremia

- Since hypertonic sodium has 513 meq/L sodium, infusion rate is then total meq divided by 513=total cc to be given, divided by the number of hours to be given over.

Hyponatremia

- Sample Case
- 38 yo WF s/p C-section with normal lytes pre-op noted to have seizures post delivery, does not respond to IV Mg, Valium. Serum sodium found to be 110. Pt weighs 60 kg.
- Meq sodium needed in first four hrs=0.5*60*8=240
- 240/513=.467 L, or 467 cc/4=117 cc/hr hypertonic saline for four hours
- Meq sodium needed in 24 hours=0.5*60*(125-110)=450
- 450/513=.877 L, or 877 cc/24=37 cc/hr.

Hyponatremia

- So after the first four hours, turn the rate down to 24 cc/hr.
- Close monitoring in ICU, check lytes q2 hrs for the first 6 hrs, then q 4 hrs.

Hyponatremia

- Chronic asymptomatic hyponatremia should be corrected much more slowly--0.5 meq/hr or 10 meq per day, whichever is less, and 18 meq in the first two days. Faster rate risks central demyelinating lesions.

Hyponatremia

- In Congestive Heart Failure
- Is caused by inability to excrete ingested water due to inadequate cardiac output and increased ADH from non-osmotic stimuli (ie, inadequate cardiac output), and in acute MI, from pain and analgesia
- Restrict fluid to 1.0 L of fluid a day or less (1.5 L is normal intake)

Hyponatremia

- In Congestive Heart Failure
- Patients presenting in this setting with hyponatremia have a poor prognosis
- ACE inhibition may be poorly tolerated with severe hyponatremia; if afterload reduction is needed, use hydralazine first, lasix, inotropic agents, to get serum sodium to at least 130 if possible

Hyponatremia

- At this point, low dose ACE inhibition may be attempted. Benefits include improving cardiac output and direct inhibition of the actions of ADH
- Low salt diet in this setting may actually contribute to hyperkalemia--Consider liberalizing salt intake if tolerated with continuation of lasix
- May not be able to achieve a normal serum sodium because the required water restriction may not be tolerated

Hyponatremia

- Demeclocycline is not recommended in this situation because of the development of nephrotoxicity, probably due to high circulating drug levels from diminished hepatic drug metabolism.

Hypernatremia

Hypernatremia

- Don't correct too rapidly either--Correct free water deficit over 48 hours
- Free water deficit=0.6 (in males)*wt(kg)*((serum sodium/140)-1), infuse half over the first 24 hrs, then the rest over 1-2 days.
- Patients are usually BOTH volume depleted and free water depleted. Remember to check the old urine electrolytes with creatinine. Correct with isotonic saline in addition to free water until FENa >1

Hypernatremia

- The above free water deficit is IN ADDITION TO!!! their ongoing MAINTENANCE free water requirements which =30 cc/kg ideal body weight.
- Fever increases these req by 15% each degree Celsius
- Tachypnea increases req by 50% for each doubling of the respiratory rate

Hyperkalemia

Hyperkalemia

- If in doubt, check to make sure it's real--but don't wait too long!
- Remember: distinguish cellular shift vs. total body overload
- Check EKG if >6.0
- If EKG changes, give calcium chloride in addition to glucose and insulin. If patient's not volume overloaded, can give sodium bicarbonate (if acidotic on ABG) or Kayexalate, 30-60 gm per dose, orally or by retention enema.

Hyperkalemia

- If they're volume overloaded and don't respond to diuretics, they need us.
- Digitalis can precipitate hyperkalemia by poisoning the Na-K ATPase pump, may need Digibind as well as dialysis (which doesn't remove digitalis products all that well, even with charcoal hemoperfusion)

Hyperkalemia

- Remember succinylcholine, Beta blockers, barium (if it leaks into blood) and Septra (the trimethoprim component acts as a potassium sparing diuretic), and of course potassium sparing diuretics can also precipitate hyperkalemia, as can penicillin antibiotics and LOW SALT DIETS! (need distal sodium delivery to excrete potassium)
- Dietary restriction usually 60 meq per day (normal intake is 80-120 meq per day)
- Think of **adrenal insufficiency** if they're hypotensive

Hyperkalemia

- Another important cause is hyperosmolar agents. This can occur with mannitol, contrast dye (rarely), and in insulin deficient patients, with glucose.
- Therefore, in the ED you should always check a fingerstick BEFORE giving the patient D50. If they're hyperglycemic to start with and insulin deficient, giving them more glucose can precipitate FATAL HYPERKALEMIA (That's a board question).

Hyperkalemia

- Check TTKG
- Remember, must check BOTH urine and serum OSMOLALITY. Urine osm must be > serum osm, and urine Na must be >20 (If it's less than 20, volume depletion alone can contribute to hyperkalemia)
- TTKG=(Urine K)/(Urine Osm/Serum Osm)/(Serum K)
- Values <6 c/w inappropriate renal potassium excretion, usually due to decreased aldosterone effect.
- Values >10 c/w non-renal hyperkalemia (excessive administration, volume depletion).

Hyperkalemia

- Heparin--a special case
- Hyperkalemia occurs in 7-8% of patients treated with heparin
- Effects start at 1-3 days and are maximal at 3-5 days, remit within 1-3 days of heparin discontinuation
- Occurs even at low doses (ie 5000 u sq BID)
- High risk patients are those with renal insufficiency, diabetes mellitus, those receiving ACE inhibitors, or receiving potassium supplementation.

Hyperkalemia

- Recommend monitoring serum potassium in anyone being given heparin for more than three days, and checking at intervals of no more than 4 days.
- If heparin must be continued and resulting hyperkalemia is significant, must DC ACE inhibitors if possible (consider change to hydralazine), restrict potassium in diet, and administer lasix or fludrocortisone (although this last would only be in extraordinary circumstances--please ask us about it first!)

Hypokalemia

Hypokalemia

- First, is it true total body depletion or just an intracellular shift?
- Shifts from adrenergic hyperactivity (MI, PE, inhaled Beta agonists, insulin overdose).
- These should be relatively short-lived, so repeat serum K in 1-2 hours.
- Check a TTKG to assess for renal potassium wasting vs. shift

Hypokalemia

- TTKG=(Urine K)/(Urine Osm/Serum Osm)/(Serum K)
- If value >4, c/w renal loss (diuretics or excessive aldosterone action), values <2 c/w GI loss or cellular shift.

Hypokalemia

- Easy enough to replace --but if they can take po, GIVE IT PO, NOT IV
- Check the magnesium too--Low magnesium can cause refractory hypokalemia
- A low salt diet may be helpful in this case. Remember, volume depletion or low salt intake impairs renal loss of potassium. Therefore, volume depletion may help in hypokalemia or even contribute to HYPERKALEMIA.

Hypokalemia

- If serum K between 3.0 and 3.5, can always replace orally.
- If serum K less than 3.0, check EKG. If abnormal (U waves, prolonged QTc) strongly consider transfer to telemetry. MUST transfer them to telemetry if on Digoxin. IV potassium replacement is justified under these circumstances.

Hypokalemia

- Remember, the maximum rate of IV potassium correction is 10 meq per hour
- UNLESS the patient is urgently unstable and you are standing by the bedside with code cart ready and looking at the monitor constantly. THEN you can give it faster, if needed.

Hypokalemia

- If you're not sure how much potassium to replace, check a timed urine (1-2 hours for IV lasix, six hours for po Lasix) for electrolytes. Multiply the concentration times the volume and you know how many meq of potassium have been lost.
- Diamox is the most potent potassium wasting diuretic, and its duration is 4-5 hours IV and 10-12 hours orally.
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Hypokalemia

- If you're really in a hurry and you think the results of a timed urine will take too long to come back, order your timed urine, record the volume, and send an aliquot as a spot sample to the lab stat.

Hypokalemia

- If hypokalemia is chronic, can use a TTKG (transtubular potassium gradient) from a spot urine: need serum OSM, urine OSM, urine lytes.
- TTKG=(Urine K)/(Urine Osm/Serum Osm)/(Serum K)
- Values >7 are c/w increased action of aldosterone, values <2 are c/w suppression of aldosterone action, and values in between are indeterminate
- Would also check 24 hr urine electrolytes
- If 24 hr urine K>30 meq d in the presence of hypokalemia, confirms inappropriate urinary potassium wasting. Give us a call!

Now, no matter where you go….

- ….you’ll be ready